Mechanisms of atelectasis in anesthesia and intensive care

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Can you tell us about your experience in regard to the relationship of lung collapse and airway closure?

Lung collapse and airway closure are common phenomena during anesthesia. The changes we see during anesthesia are also seen in the ICU, but much more pronounced.

The FRC is reduced by about 20% during anesthesia. The interesting point is that when you are upright, you may have an FRC of about 3 liters, depending on body size. If you lie supine, it will be reduced by about 0.7 or 0.8 liters. Abdominal organs push up the diaphragm, which reduces the FRC. And then we are going to anesthetize, which will bring it down to about 2 liters, which means we are close to the residual volume. Residual volume is the volume you have in the lungs at maximum expiration. It is important to realize this simple fact: when we are treating patients during anesthesia in the supine position, the lung volume is at residual volume.
Can that have an effect on the patency of the lung tissue?

That is what we asked 20-25 years ago. In the CT scans of the anesthetized subject, it is evident that the diaphragm and liver have moved cranially, and consequently reduced FRC. The major cause of reduced FRC in anesthesia is cranial displacement of the diaphragm.

We found something interesting at the bottom of those lung CT scans that looked like a “roaring sea”. We conducted animal experiments, and CT scans showed a large amount of densities. Some animals have severe hypoxemia in the supine position. When we removed the lungs of some of the animals and made a histological assessment, we found normal tissue and some completely collapsed tissue, with a very sharp border. This corresponds to atelectasis, with some edema and congestion. When we presented this for the first time, it was a new discovery.

So we established that the dense area was atelectasis, and we wondered whether it was due to anesthesia or something else. We moved on to patients who were awake and spontaneously breathing, and found no densities in the CT. We thereafter induced anesthesia in these patients with a face mask, under spontaneous breathing, and found the densities, or atelectasis, appearing almost immediately. When we paralyzed the patients, the densities increased. We know that the anesthetic is the cause of atelectasis, and that we do not need to paralyze a patient to induce atelectasis.

What else was established in these first studies with regard to atelectasis?

In post-operative CT scans of these patients, we found that these densities, or atelectasis, remain for one hour after anesthesia, or even for several days. You may see a slow decrease in atelectasis over time, but more than a quarter may still have atelectasis on the fifth day post surgery.

From these first studies we concluded that atelectasis during anesthesia is found during spontaneous breathing and artificial ventilation, with intravenous and inhalational anesthetics, and that the atelectasis will last a few days post-operatively, and may cause post-op pulmonary complications. Loss of muscle tone is a prerequisite for developing atelectasis.

We could also conclude that about 90% of all patients will develop atelectasis during anesthesia, and that 3-4% of lung area (basal) is affected, with at least 10-15% of lung tissue involved. This cannot be seen on an X-ray. In uneventful anesthesia, it’s standard to leave the patient with at least 10-15% of the lung collapsed.

After thoracic surgery, atelectasis is much more pronounced. Over 40% of lung tissue is collapsed, with a very slow reopening of the tissue. Most of the atelectasis is near the diaphragm.

What in your opinion is the relationship of shunt in atelectasis?

Shunt (perfusion of the lung that is not oxygenated) is nearly always caused by atelectasis in the anesthetized, otherwise lung-healthy subject. This is also the major cause of acute respiratory failure in the intensive care setting, but there, another cause of collapse can be fluid filling of the alveoli. But shunt is the major cause of hypoxemia, or impeded oxygenation, in both cases.

Shunt was established to have a direct correlation to atelectasis in anesthesia. We also found that it is related to intensive care. We did a CT and gamma camera SPECT study in patients, with CT scan and vertical distribution of ventilation and perfusion in the same lung segment. We found atelectasis in the bottom of both lungs, and could see how the ventilation and perfusion were distributed in the lung, from top to bottom.

The perfusion increased down the lung – a common distribution pattern when the patient is awake, during anesthesia, and in the ICU, provided you do not have a vascular abnormality. There was a slight decrease in perfusion at the bottom of the lungs.

In the healthy and awake subjects, ventilation also increases along the lung, similar to the perfusion. But in the anesthetized subject, we have a very different pattern. Most of the ventilation goes to the upper half, so there is a clear mismatch. In the upper half, ventilation is in excess of perfusion. In the lower half, we have less ventilation than perfusion. And at the bottom, we have no ventilation at all. This corresponds to the atelectasis.

So why do we have a decrease in ventilation in a zone above the atelectasis? That is due to airway closure. When you exhale, you can get closure of air ducts, a normal phenomenon. It is lung volume dependent. In healthy adult subjects, sitting up, there will be no airway closure during normal breathing. But if they lie down, they will suffer from airway closure as lung volume decreases. During anesthesia the further decrease in lung volume caused airway closure in all subjects above 30 years of age. If airways remain closed all the time, the gas in closed regions will be absorbed. The airways that open for inspiration and close for expiration will allow for a certain level of gas exchange, but it is reduced. And that can explain the decrease of ventilation in the zone above the atelectasis.

An important issue for many physicians is how to manage airway closure with regard to aeration and recruitment. Do the airways have an important physiological role that is not always fully appreciated?

Yes, they do indeed. In fact, we should remember that airway closure is a normal phenomenon, which is more obvious or dominating if lung volume is reduced. When lung volume is reduced, alveoli decrease in size, and the airways do as well. The decrease in lung volume, with the subsequent decrease in airway caliber, promotes airway closure. So during anesthesia, or in intensive care when using muscle paralysis or sedatives, you reduce or eliminate respiratory and other muscle tone. Then lung volume is reduced, and you provoke or promote airway closure. So this is a normal phenomenon made worse by the decrease in lung volume.

Airway closure was first demonstrated in the mid-60s. People who know about airway closure have thought that it is something that occurs in diseased conditions. It was proposed as a measure of early obstructive lung disease before any other sign of the disease was existent. For some reason, people have disregarded the fact that it is a normal phenomenon with impact on oxygenation of lungs. In fact with age, oxygenation of blood is impaired; PaO2 successively decreases as we get older. The explanation is airway closure.

Airway closure and atelectasis explain as much as three-quarters of oxygen impairment during anesthesia.

What can we do about airway closure and atelectasis?

In regard to airway closure, we can elevate FRC by means of PEEP. In regard to atelectasis there are a number of possibilities: give PEEP, increase muscle tone, “sigh” the patient, and avoid high O2 concentrations (FiO2), as this can affect the absorption of gas.

What happens to the lung physiologically when PEEP is administered or discontinued?

CT studies show that in patients with large atelectasis (>25% of lung tissue) giving PEEP
of 10 cm of water almost eliminates the atelectasis. If PEEP is discontinued all the atelectasis returns, so there is no lasting effect. When we discontinue the PEEP for any reason, such as suctioning, the atelectasis will return within a minute.

We learned about the lung physiological effects of PEEP by looking at the perfusion distribution to anesthetized patients with gamma camera technique. We could see that at 10 cm of water, PEEP redistributes with more perfusion going to the lower lung. If we have atelectasis in the lower portion, we get shunt with ZEEP. We can also get shunt with PEEP even if the atelectasis is reduced, since the perfusion has been forced down. In general, we do not see an improvement in PaO₂ by applying a standard PEEP in anesthesia.

As a routine tool, PEEP will not improve oxygenation. Hewlett et al. in 1974 concluded that there was no place for the indiscriminate use of PEEP in routine anesthesia. We can conclude that PEEP has no effect on PaO₂ in unselected patients, has no remaining effect after discontinuation, and decreases the cardiac output.

What about the use of PEEP in the intensive care setting?

Yes, you can recruit lung tissue that has collapsed by increasing airway pressure. When you apply a PEEP, you have to increase the inspiratory pressure in order to insufflate the air and gas into the lungs. The increased airway pressure required when applying PEEP will help open up atelectatic regions. Strictly speaking, it is not the PEEP per se that recruits alveoli; it is the airway pressure, or pressure above PEEP that is responsible for the recruitment phase. The PEEP will prevent re-collapse. The problem with PEEP is that there is no lasting effect. As soon as it is discontinued, the alveoli will collapse again within a minute. Many people might not yet realize this fact. So when you give PEEP and think that you have opened up the lung, but stop using it, the lung will collapse. Another thing with PEEP is that it will affect the distribution of blood flow in an unfavorable way. It might be needed in order to keep the lung open, but one should realize that PEEP is not the ultimate tool. It is a tool that may be valuable in the intensive care setting, but it may not have the same value in anesthesia.

In contrast to PEEP, what we call vital capacity maneuvers (lung recruitment procedures) do have a lasting effect. We may ask why one technique does not have a long lasting effect when another one does. With PEEP, we open up unstable alveoli, but the alveoli remain unstable and need continuous support by means of airway pressure. The difference with vital capacity maneuvers is that they restore the stability of the alveoli.

When a low compliance lung is treated with a vital capacity maneuver, the alveoli become stable because surfactant spreads on the alveoli surface. To get this distribution of surfactant, you need a vital capacity maneuver, at a higher airway pressure compared to a normal size tidal breath, which is not enough to open up collapsed areas and generate new surfactant.

What effects do increasing muscle tone or “sighing” the patient have on atelectasis?

In regard to preserving or increasing muscle tone, it has been demonstrated by Tokics et al. that ketamine prevents atelectasis but if paralysis is added, collapse occurs as with other anesthetics. We also determined that phrenic nerve stimulation that tenses the diaphragm reduces atelectasis, but it is very difficult to carry out.

In regarding to “sighing” the patient as a recruitment maneuver, we established early in anesthesia, with lung healthy patients in CT scans, that atelectasis was present at atmospheric pressure. We then inflated the lung at a pressure of 10 cm H₂O, and nothing happened to the atelectasis. We inflated the lung with 20 cm H₂O, and nothing happened. The atelectasis was similar to how it was at atmospheric pressure. So what I was taught 40 years ago, to give a double tidal volume now and then, has no effect. You do not open any atelectatic tissue by inflating the lung with 20 cm H₂O in healthy subjects. However, this is not the case in ARDS patients, where you can open up tissue! We finally opened up the lung at 40 cm H₂O. This will have a lasting effect, as established by Rothen in 1993. But 40 cm of airway pressure during a longer period of time can stop the heart. We have found that eight or nine seconds is enough to open what you may be able to recruit.

“Sighing” the patient requires a vital capacity maneuver. One can ask about the risks of barotrauma, volutrauma, and the short-term decrease in cardiac output.

What is your experience in research of the use of 100% oxygen?

In patients who are ventilated at 100% oxygen after recruitment maneuvers, there are no lasting effects. Oxygen concentration has a very strong influence on atelectasis. We can conclude that a vital capacity maneuver is efficient with a moderate FiO₂ but with 100% O₂, atelectasis will return within five minutes of ventilation. This leads to the question of whether pre-oxygenation at 3-4 minutes during anesthesia induction causes atelectasis, and whether we should avoid pre-oxygenation.

We looked at a group of patients whose pre-oxygenation was limited to 30% oxygen. When they were awake there was no atelectasis. When they were anesthetized, there was no atelectasis either. At 30 minutes of anesthesia, there was a tiny amount of atelectasis. If you look at a time sequence, you see that 30% oxygen is associated with very little atelectasis over time. So we have learned to avoid a high FiO₂ during anesthesia induction and during anesthesia, but to balance this benefit with the risk of hypoxemia.

In a recent study by Edmark et al., anesthesia was induced in patient groups with different O₂ concentrations. One group received 100% O₂, one group received 80% O₂, and the third group received 60% O₂. The groups with 60% and 80% pre-oxygenation were associated with very little atelectasis. But the 100% group was associated with broad levels of atelectasis. Apnea tolerance was also studied in the same groups, and it may be said that pre-oxygenation at 80% O₂ will not be too dangerous for these patients.

The benefit of lowering O₂ concentration during the induction of anesthesia is that there is less atelectasis, and recruitment maneuvers may be used afterward to retain this effect. Magnusson et al. established that post-oxygenation after surgery and before extubation may have an effect on atelectasis.

The mechanisms of atelectasis during anesthesia are loss of muscle tone (fall in FRC, increase in airway closure), high inspired oxygen concentration (> 80%), and impaired surfactant function.

The reason why atelectasis reappears so fast when we discontinue PEEP must be explained by surfactant impairment. Collapsed alveoli mean that the surfactant will be destroyed. If we open up the alveoli, with additional PEEP the surfactant function is not restored, and the alveoli remain unstable. The combination of these factors sets off a rapid chain of events that leads to atelectasis in just minutes.

However, if we make a vital capacity maneuver, surfactant is released from its production sites and delivered to the alveolar wall and bronchi. The vital capacity maneuver restores surfactant function, and the alveoli remain stable.
How does this situation in anesthetized patients differ from ARDS patients in the ICU?

Collapse in the ARDS situation differs somewhat. Not only is there atelectasis, but also fluids that fill the lungs.Gattinoni studied these patients with CT, and found densities in the lower parts of the lung. Neumann et al. studied the effect of breathing on atelectasis in ARDS patients, and established that there is considerable collapse with certain PEEP levels in the models. PEEP of 20 or 25 is necessary to reduce the cycling collapse. In ARDS patients, a certain amount of atelectasis and fluids can be seen at end-expiration. At inspiration we will open up some of them, but there is further collapse at the next expiration. This cyclic collapse may be more harmful to the lung than the continuous collapse that the other parts of the lung may be exposed to, or even than over distension of tissue that is aerated all the time.

With regard to atelectasis, can we modify the ventilatory support by adding spontaneous breathing?

I am interested in that, since we see such differences between the mechanically ventilated and spontaneously breathing healthy lung. Christian Putensen in the early-to-mid-90s performed Airway Pressure Release Ventilation (APRV) experiments using dogs. In mechanical ventilation, the PaO<sub>2</sub> increased when spontaneous breathing was present. Shunt was also reduced in these subjects. Putensen and colleagues also established that in human patients on APRV, PaO<sub>2</sub> can be improved in the presence of spontaneous breathing with reduced shunt as well.

How do spontaneous breaths improve gas exchange, and what are the effects on lung volume, atelectasis, ventilation and perfusion?

There is a beneficial effect on aeration, as demonstrated in studies with CT scans. There is also an effect of the APRV on ventilation distribution. In a gamma camera study by Neumann et al., from diaphragm to apex, and from posterior to anterior, the ventilation was shown in more apical and anterior regions. With APRV, we are able to distribute some ventilation to other regions, where we have more perfusion.

Thoracic EIT (Electrical Impedance Tomography) has also been used to show how ventilation is distributed and the effects of respiratory modes on regional ventilation.

In Pressure Control, most of the ventilation goes to the upper region. In APRV there is ventilation in the upper part, which is being mechanically ventilated, but more in the lower part where spontaneous breathing is evident. If we switch to CPAP, there is mainly spontaneous breathing in the lower part. So you can see how it varies in regard to distribution of the spontaneous breath and the mechanical breath.

Why do we have these differences?

When you have a relaxed diaphragm during mechanical ventilation, there is a certain amount of pathology and atelectasis consolidation. When we inflate the lung by increasing airway pressure, we push away the diaphragm and elevate the rib cage to some extent. The displacement of the diaphragm will be mainly in the upper part of the diaphragm because the pressure in the upper, anterior part of the abdomen is lower. If we say that we have an edematous patient with ascites and fluid, the pressure will increase along the abdomen, corresponding to 1 cm H<sub>2</sub>O to 1 cm of distance. If there is a 20 cm distance, there will be a 20 cm higher pressure in the lower part than in the upper part. Therefore it is easier to push away the diaphragm in the upper part than in the lower part. So we get a preferential displacement of the diaphragm anteriorly in the supine position.

Now with spontaneous breathing, we have a completely different displacement. Most of the movement is in the dorsal part. This is because the diaphragm in this situation becomes an active muscle, moving itself. We have more muscle fibers in the dorsal section, and an elongation of the fibers in this area compared to the anterior part. This elongation makes the fibers stronger. The muscle is stronger and moves more forcefully in the dorsal part than the anterior part, during an active spontaneous breath.
In synchronized ventilatory support, what is the best way to trigger the ventilator, by flow, pressure or EMG?

This is an area of interest. Despite some of the benefits of APRV, I don’t think it is the perfect tool. This sustained non-synchronized pattern of behavior cannot be ideal. If we look at the triggering of the ventilator, Martin Tobin et al. demonstrated that diaphragm contraction causes muscle contraction after some time, which lowers airway pressure. But there is a time lapse until the pressure has dropped enough in the ventilator to trigger a breath. So from when the EMG signal is transmitted from the brain, there is a delay of half a second or more before the ventilator provides a breath to the patient.

In recent times, Sinderby et al. have researched the use of a catheter to enable the recording of the diaphragmatic excitation, EMG signal is transmitted from the brain, there could be a shortcut with less delay. The signal is also proportional to the demand of the patient. So we can use the magnitude of the signal to determine the tidal volume. If this could be made into a clinically usable tool, it could be very useful because you would have a type of pressure support that is totally controlled by the patient, provided he or she is delivering a signal from the diaphragm.

Biography
Göran Hedenstierna was named Professor in Clinical Physiology at Uppsala University in 1988, and held the position of Chairman of Clinical Physiology for 14 years. He has been Chairman of the Department of Nuclear Medicine at Uppsala University Hospital since 2002. He has also been visiting or honorary professor at numerous university institutions in the US, France, China and Italy.

He has published over 430 scientific papers and reviews, primarily in his scientific research areas of atelectasis and gas exchange, lung edema and nitric oxide. He holds a number of scientific committee positions as chairman or member in institutions such as the Swedish Research Council, Swedish Heart and Lung Foundation, the Fleischer Society, the Royal College of Anaesthetists and the German Society of Anaesthetists.


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